

Diagnostic approach to children and adolescents with short stature

Author

Alan D Rogol, MD, PhD

Section Editor

Mitchell Geffner, MD

Deputy Editor

Alison G Hoppin, MD

Contributor disclosures

All topics are updated as new evidence becomes available and our [peer review process](#) is complete.

Literature review current through: Mar 2016. | **This topic last updated:** Sep 11, 2014.

INTRODUCTION — Short stature is defined as a height that is 2 standard deviations (SD) or more below the mean height for individuals of the same sex and chronologic age in a given population. This translates to a height that is below the 2.3rd percentile.

The most common causes of short stature beyond the first year or two of life are familial (genetic) short stature and constitutional short stature (also known as constitutional delay of growth and puberty), which are normal nonpathologic variants of growth (see "[Causes of short stature](#)", [section on 'Normal variants of growth'](#)). The goal of the evaluation of a child with short stature is to identify the subset of children with pathologic causes, such as Turner syndrome, inflammatory bowel disease or other underlying systemic disease, or hormonal abnormality. The evaluation also assesses the severity of the short stature and likely growth trajectory, to facilitate decisions about intervention, if appropriate. Some components of the evaluation can reasonably be performed in the primary care setting, including initial interpretation of the growth chart and growth potential (based on measured heights of the child's parents), calculation of height velocity (HV), initial laboratory screening for an underlying systemic or endocrine disease, if suspected based on symptoms. If HV is slow, then bone age determination should be performed if expert interpretation is available. Other components of the evaluation, including review of the bone age results and the detailed evaluation for causes of short stature, are typically performed by a pediatric endocrinologist, if available.

Referral patterns reveal substantial sex differences in the evaluation and treatment of short stature [1-4]. Boys are referred for evaluation more often, at younger ages and for less severe height deficits as compared with girls. As an example, in one retrospective review of 288 children referred to a single center for assessment of short stature, the male:female ratio was 1.9:1 [1]. At the time of referral, the height deficit was significantly greater for girls than boys (median height Z-score, -2.4 versus -1.9), and organic disease was more common among girls (40 versus 15 percent). Similarly, studies of growth hormone registries have shown preferential treatment of boys compared with girls with an approximate ratio of 2:1 [2,3].

This apparent gender bias may be due to under-appreciation of growth problems in girls, leading to fewer evaluations of girls for short stature. Alternatively, it may be due to increased societal pressure for tall stature in boys, leading to increased referral and growth hormone treatment of boys without organic causes of short stature. These findings emphasize the need for accurate growth monitoring during the health care maintenance of all children to ensure appropriate referral and treatment.

This topic will review the diagnostic approach to children with short stature, beginning with a brief review of normal growth and development. The causes of short stature are discussed separately. (See "[Causes of short stature](#)".)

NORMAL GROWTH — Serial measurements of growth are an important parameter in monitoring the health of children. In general, a normal pattern of growth suggests good health, while growth that is slower than normal raises the possibility of an underlying subacute or chronic illness, including an endocrinologic cause of growth failure.

Statural growth is a continuous but not linear process. There are three phases of postnatal growth (infantile, childhood, and pubertal), each of which has a distinctive pattern [5]. The phases are similar for boys and girls, but the timing and pace of growth differ, particularly during puberty.

- Intrauterine – Size at birth is determined more by maternal nutrition and intrauterine and placental factors than by genetic makeup. Not all the genes that affect growth may be expressed at birth. As a result, the correlation coefficient between birth length and adult height is only 0.25 [6].
- Infancy – During the first two years of life (infantile phase), linear growth initially is very rapid and gradually decelerates. Overall growth during this period is about 30 to 35 cm. The length (and weight) of premature infants must be corrected for gestational age, at least for the first year. However, growth is often accelerated during the second half of the first year in otherwise healthy children born early.

An infant's height curve often crosses percentile lines during the first 24 months of life as the growth moves away from the influences of the intrauterine environment and toward the child's genetic potential. As examples, infants born small because of placental or uterine constraint may move upwards across percentile curves ("channeling up"), whereas infants born large because of maternal-fetal overnutrition may cross downward across percentile curves ("channeling down"). After this adjustment period, the correlation between length at two years and adult height is 0.80 [6].

- Childhood – The childhood phase is characterized by growth at a relatively constant velocity, with some slowing in later childhood. Most children grow at the following rates (representing the 10th to 90th percentiles):
 - Age two to four years: 5.5 to 9 cm/year (2.2 to 3.5 inches/year)
 - Age four to six years: 5 to 8.5 cm/year (2 to 3.3 inches/year)
 - Age six years to puberty:
 - 4 to 6 cm/year for boys (1.6 to 2.4 inches/year)
 - 4.5 to 6.5 cm/year for girls (1.8 to 2.6 inches/year)
- Adolescence – The pubertal phase is characterized by a growth spurt of 8 to 14 cm per year due to the synergistic effects of increasing gonadal steroids and growth hormone [7]. In girls, the pubertal growth spurt typically starts around age 10, but may start as early as age 8 for early maturing girls. In boys, the pubertal growth spurt typically starts around age 12, but may start as early as age 10 in early maturing boys [8].

The "rule of fives" incorporates these typical phases of growth and provides an estimate for normal height and growth velocity in a given age group, as shown in the table (figure 1). Actual height and growth rate in a healthy child can vary substantially around these approximations.

EVALUATION OF GROWTH — Evaluation of a child suspected of having short stature is guided by answering the following questions. Although the causes and clinical presentation of short stature vary by age group, the same questions are relevant for children of any age:

- How short is the child?
- Is the child's height velocity (HV) impaired?
- What is the child's likely adult height?

The answers to these questions guide a focused history and physical examination, and in some cases laboratory evaluation, to determine the cause and appropriate management of the child with short stature ([algorithm 1](#)).

Is the child short? — Accurate measurement of length or height is essential; inaccurate measurements or aberrant plotting is one of the more common causes of apparent growth failure. Length is measured lying down and should be used for infants and children up to 24 months of age ([figure 2A-B](#)); height is measured standing and should be used for children two years and older (if possible) ([figure 3](#) and [figure 4A-B](#)). The results should be plotted on the growth chart that is appropriate for the child's age and gender. Weight should also be measured to provide additional information about the child's nutritional status. (See "[Measurement of growth in children](#)".)

Percentiles and Z-scores can be calculated using a calculator for recumbent length ([calculator 1](#)), or standing height for boys ([calculator 2](#)) or for girls ([calculator 3](#)). For the purposes of an endocrine evaluation, short stature is defined as a length or height more than 2 standard deviations (SD) below the mean (ie, a Z-score <-2), which corresponds to a height that is $<2.3^{\text{rd}}$ percentile.

- Height above the 2.3^{rd} percentile (>-2 SD) – These children generally do not require further specific evaluation unless there are additional reasons for concern, such as progressively decreasing height percentiles (implying growth failure), dysmorphic features, or evidence of underlying systemic disease.
- Height below the 2.3^{rd} percentile (≤-2 SD) – These children have short stature and should undergo a more detailed evaluation, starting with evaluation of growth rate, as outlined in the following sections. The first steps of this evaluation usually can be performed in the primary care setting. Early referral to a specialist is appropriate if the growth rate is very slow.
- Height less than the 1^{st} percentile (≤-2.25 SD) – These children have extreme short stature and usually should be referred to an appropriate subspecialist for a detailed evaluation, where available. The first steps in the evaluation will be similar, but a higher index of suspicion for pathologic causes of growth failure is appropriate.

Is the child's height velocity impaired? — Determination of the child's growth rate, or HV, is an essential component of the evaluation for short stature and can be considered a "vital sign" ([algorithm 1](#)). This requires serial measurements of height, which should be measured along with weight at each well-child visit. Serial measurements help to determine whether the child's growth rate is within normal ranges and whether it progressively deviates from its previous growth channel (or percentile curve). The HV should be calculated (in cm/year) using accurate measurements of height and an interval between measurements of at least six months.

For children two years and older, **growth failure is likely** if:

- The height-for-age curve has deviated downwards across two major height percentile curves (eg, from above the 25th percentile to below the 10th percentile).
- Or, if the child is growing slower than the following rates:
 - Age two to four years: HV less than 5.5 cm/year (<2.2 inches/year)
 - Age four to six years: HV less than 5 cm/year (<2 inches/year)
 - Age six years to puberty:
 - HV less than 4 cm/year for boys (<1.6 inches/year)
 - HV less than 4.5 cm/year for girls (<1.8 inches/year)

Short children with HV **above** these cut points usually have a nonpathologic cause of short stature, such as familial short stature or constitutional delay of growth. For these children, a basic evaluation for short stature usually is sufficient, consisting of a focused history, physical examination, adult height prediction, and bone age determination, as outlined in the following sections [9]. (See '[Prediction of adult height](#)' below and '[Bone age determination](#)' below.)

Short children with HV **below** these cut points are more likely to have a pathologic cause of short stature and warrant additional attention. In addition to the basic evaluation outlined above, laboratory screening for pathologic causes of growth failure is warranted. The clinician should be particularly alert for subtle symptoms of underlying systemic disease (eg, Crohn disease) and for evidence of Turner syndrome in girls [9]. The detailed evaluation may be most appropriately performed by a pediatric endocrinologist, if available. (See '[Laboratory and imaging studies](#)' below.)

Short children with HV **substantially below** these cut points are most likely to have a pathologic cause of short stature and should undergo a detailed evaluation for pathologic causes of growth failure by a pediatric subspecialist, if available.

Alternatively, for a more precise assessment, the child's HV can be plotted on an HV chart ([figure 5A-B](#)) to determine the HV percentile (or SD [Z-score]) for the child's age and gender (note that this is different from the height-for-age percentile). In general, HV between the 10th and 25th percentile should raise concern for possible growth failure, and an HV below the 10th percentile warrants a thorough evaluation for growth failure.

Prediction of adult height — Adult height is determined by a combination of genetic potential and many other factors that influence somatic growth and biologic maturation. No method accurately predicts adult height, and there is wide variation in predicted adult height among the different methods. However, an estimate of adult height can be developed using information about heights in the biologic family, combined with information about the child's own growth and level of skeletal development. The results help to guide decisions about evaluation and treatment, and also provide some information about the possible causes of short stature in a particular patient.

Is the child's growth within the range for the family? — The next step is to determine the height range expected for the child's biologic family and compare it with the child's growth trajectory. In most populations, a predicted adult height that is <63 inches (160 cm) for men and <59 inches (150 cm) for women is considered short stature, corresponding to more than 2 SD below the mean (<2.3rd percentile) for adults of the same population and gender.

- Midparental height** – An estimate of a child's genetic height potential can be obtained by calculation of the midparental height, which is based upon the heights of both parents and adjusted for the sex of the child ([algorithm 1](#)). This estimate is also known as the "target height". Whenever possible, the parents' heights should be directly measured rather than

self-reported. The calculation can be performed using a calculator ([calculator 4](#)) or the following equation:

- For girls, 13 cm (or 5 inches) is subtracted from the father's height and averaged with the mother's height.
 - For boys, 13 cm (or 5 inches) is added to the mother's height and averaged with the father's height.
 - For both girls and boys, 8.5 cm (2.4 inches) on either side of this calculated value (target height) represents the 3rd to 97th percentiles for anticipated adult height [10].
- In this calculation, the 13 cm (or 5 inches) represent the average difference in height of adult men and women; thus, the child grows, on average, to the midparental height percentile.

●**Projected height** – The projected height for a child older than two years is determined by extrapolating the child's growth along the current channel to the 18- to 20-year mark ([figure 6](#)). If the child's bone age is delayed or advanced, then the projected height should be plotted based on the bone age rather than the chronologic age. (See '[Bone age determination](#)' below.)

The child's projected height is then compared with the range calculated from the midparental (target) height.

- If the child's projected height is within 8.5 cm (2 SD) of the midparental height, then the child's height is within the expected range for the family. This child probably has familial short stature, which is considered a variant of normal growth. (See "[Causes of short stature](#)", [section on 'Familial short stature'](#).)
- If the projected height is more than 8.5 cm (2 SD) below the midparental height, then the child can be considered abnormally short for his or her family.

Is there evidence of delayed or accelerated growth? — Most children with severe short stature (height <-2.25 SD) or growth failure should undergo a radiographic determination of bone age. This helps to determine whether the child's growth is delayed or accelerated compared with his or her chronological age.

Bone age determination — Bone age (also known as skeletal age) is typically determined from a radiograph of the left hand and wrist, and requires expert interpretation. The methods used most commonly for determining skeletal age are the Greulich and Pyle Atlas [11] and the Tanner-Whitehouse (TW2) method [12]. The results inform estimates of the child's growth potential and likely adult height, as follows:

- Delayed or advanced bone age is defined as a bone age that is 2 SD or more below or above the mean, respectively. This is approximately 20 percent below or above the chronological age. This translates to a difference between bone age and chronological age of approximately 12 months between 2 and 4 years of chronological age, 18 months between 4 and 12 years, and 24 months after age 12 ([figure 7A-B](#)). If the bone age is delayed or advanced near or beyond these parameters, then the projected height should be recalculated based on the bone age rather than the chronologic age. This will provide a more accurate assessment of projected height. As an example, if an eight-year-old boy is 117 cm tall and has a bone age of 6.5 years, this corresponds to the 3rd percentile for chronological age, but to the 35th percentile for skeletal age, suggesting that the child may have constitutional delay of growth.
- The bone age can be used to predict the child's adult height. The technique developed by Bayley-Pinneau is most commonly used for children approximately six years and older [13].

This technique employs a table to translate the child's bone age and chronological age to a decimal fraction of adult height (eg, 0.75). To predict adult height, the present height is divided by the fraction of adult height.

Other methods use different algorithms to predict adult height from height measurements and bone age data, and include the Tanner-Whitehouse II (TWII), Roche-Wainer-Thissen (RWT), and Khamis-Roche (KR) methods. RWT and KR incorporate midparental height in addition to bone age, and TWII uses a different measure of bone age. Comparison of these methods reveals a wide range of adult height predictions. Overall, the TWII method tends to under-predict adult height, while the BP and RWT methods tend to over-predict adult height in boys [14]. The BP method is most likely to identify a short child as a candidate for growth hormone therapy [15]. Methods that incorporate bone age are generally more accurate in predicting adult height than the simple midparental height method. However, there is a wide variation in height prediction, and the same method may yield significantly different adult height predictions at different ages [14].

The results of the bone age determination also provide important information about possible causes of the short stature ([algorithm 1](#)):

- A significantly delayed bone age is consistent with constitutional delay of growth and puberty (CDGP), which is considered a variant of normal growth. However, significantly delayed bone age is also seen in many types of pathologic growth failure, including nutritional deficiency, underlying systemic disease (such as inflammatory bowel disease), and growth hormone deficiency. The HV helps to distinguish between these categories: children with CDGP tend to have normal or low-normal HV until they reach bone age of 11 years in girls, or 13 years in boys. By contrast, children with underlying systemic or endocrine disease tend to have progressive decreases in HV. Children with significantly delayed bone age should undergo a focused history and physical examination to evaluate for symptoms and signs of systemic or endocrine disease (see '[Additional evaluation for causes of short stature](#)' below). Their growth should also be carefully monitored.
- A normal bone age is consistent with several diagnostic possibilities: in a child with short parents, a normal bone age supports the diagnosis of familial short stature. However, a normal bone age is also seen in younger girls with Turner syndrome. Moreover, bone age may be only mildly delayed in early or mild forms of some of the systemic diseases that cause growth failure. Therefore, a bone age that is within normal limits suggests that underlying genetic or systemic disease is unlikely, but not impossible.
- Advanced bone age is occasionally seen in older children and adolescents with short stature, especially precocious puberty and hyperthyroidism. These children usually experienced accelerated early growth but are at risk for early epiphyseal closure, resulting in short stature as an adult, if not properly diagnosed and treated. (See "[Causes of short stature](#)", section on '[Sexual precocity](#)'.)

ADDITIONAL EVALUATION FOR CAUSES OF SHORT STATURE — In addition to the evaluation of growth outlined above, a focused history and physical examination contribute information that helps to categorize the cause of short stature ([table 1](#) and [algorithm 1](#)). The key information is organized here according to the diagnostic category of the short stature. Further

details about each cause of short stature are given in the linked topic review. (See "[Causes of short stature](#)".)

Are there features that suggest that this is a normal variant of short stature? — The two most common causes of short stature are familial (genetic) short stature and constitutional delay of growth and puberty (CDGP, also termed constitutional short stature for prepubertal children). These growth patterns often can be distinguished from one another, but some children have features of both ([table 2](#)). Unless there are signs or symptoms of underlying disease, children with these growth patterns usually require only a basic evaluation including a focused history, physical examination, and bone age determination. (See '[Laboratory and imaging studies](#)' below.)

- In familial short stature, height velocity (HV) and bone age are within the normal range and one or both parents are short. (See "[Causes of short stature](#)", section on '[Familial short stature](#)'.)
- In CDGP, the child's growth rate is appropriate for his or her bone age, which is delayed compared with chronological age. If the height is plotted using the bone age rather than chronological age, the projected height is within the range predicted for the family and often within the normal range for the population (ie, adult height that is ≥ 63 inches [160 cm] for men and ≥ 59 inches [150 cm] for women). There is often a family history of delayed growth and/or puberty (a parent who was a "late bloomer"). (See "[Causes of short stature](#)", section on '[Constitutional delay of growth and puberty](#)'.)

Are there features suggesting pathologic growth failure? — The history, review of systems, and physical examination should include the following elements to assess for a variety of pathologic causes of short stature ([table 1](#) and [algorithm 1](#)). The findings may influence selection of laboratory tests and/or imaging.

Features suggesting underlying systemic disease — A variety of systemic diseases are associated with attenuated growth during childhood, usually with delayed bone age. This is particularly true for inflammatory disease processes (such as Crohn disease or juvenile idiopathic arthritis [JIA]), diseases that cause malabsorption or malnutrition, or those that increase energy needs (cardiac or immune defects). Therefore, a complete medical history and review of systems is important to the assessment of short stature.

Key elements of the history include (see "[Causes of short stature](#)", section on '[Systemic disorders with secondary effects on growth](#)'):

- Gastrointestinal symptoms, including appetite, abdominal pain, diarrhea, and rectal bleeding – Suggests the possibility of Crohn disease or celiac disease.
- Pulmonary symptoms, including severe asthma, recurrent infections – Suggests the possibility of cystic fibrosis or immunodeficiency.
- Recurrent infections – Suggests the possibility of immunodeficiency; recurrent otitis media with the need for myringotomy tubes is associated with Turner syndrome.
- Arthralgias or arthritis – Consistent with inflammatory bowel disease, rheumatic diseases (eg, JIA), or celiac disease.
- Medications – Prolonged or frequent use of glucocorticoids (including inhaled glucocorticoids) may contribute to growth failure (see "[Causes of short stature](#)", section on '[Glucocorticoid therapy](#)'). Use of stimulants for attention deficit disorder (ADD) also has been associated with mild delay in growth, although this effect usually is transient. (See "[Causes of short stature](#)", section on '[Systemic disorders with secondary effects on](#)

[growth'](#) and "[Pharmacology of drugs used to treat attention deficit hyperactivity disorder in children and adolescents](#)", section on 'General adverse effects'.)

Key elements of the physical examination include:

- Weight loss, poor weight gain, underweight-for-height, and/or delayed puberty – These findings are consistent with many underlying systemic diseases, psychosocial deprivation, or food restriction. By contrast, most endocrine causes of short stature are associated with overweight-for-height.
- Oral ulcers or large anal skin tags – These findings are common in Crohn disease and may be the presenting symptoms.

Features suggesting genetic or endocrine disease — Endocrine disorders are relatively uncommon causes of short stature but important to diagnose because they respond to specific treatment. These endocrine disorders are often characterized by delayed bone age. An exception is precocious puberty, in which accelerated early growth may be accompanied by early epiphyseal maturation and adult short stature.

Key elements of the history relevant to endocrine disease include (see "[Causes of short stature](#)", section on '[Endocrine causes of growth failure](#)'):)

- Sluggishness, lethargy, cold intolerance, constipation – These symptoms suggest hypothyroidism.
- Developmental delay/learning disabilities – Problems with nonverbal learning skills are common in Turner syndrome. Developmental delay is common in Noonan or Russell-Silver syndrome, and in pseudohypoparathyroidism. Acquired hypothyroidism is often associated with altered school performance. Many syndromes with developmental delay also include short stature, such as Down, Prader-Willi, and Bloom syndromes.
- Neuropsychological changes – Symptoms of psychiatric disease occur in over one-half of patients with Cushing's syndrome of any etiology.

Key elements of the physical examination include:

- Increased weight-for-height – Obesity is nearly universal in Cushing's syndrome (with central fat distribution). Increased weight-for-height is also consistent with hypothyroidism, growth hormone deficiency, or pseudohypoparathyroidism.
- Facial dysmorphism
 - Hypertelorism, downward eye slant, low-set ears – Noonan syndrome
 - Prominent forehead, triangular face, downturned corners of the mouth – Russell-Silver syndrome
 - Midface hypoplasia, frontal bossing – Achondroplasia
 - Midline defects – Associated with hypothalamic-pituitary hormone deficiencies
- Optic discs – Papilledema suggests a central nervous system mass effect. Optic nerve hypoplasia suggests septo-optic dysplasia, which is associated with pituitary hormone deficiencies. In optic nerve hypoplasia, the optic disc is small and often pale and surrounded by a yellowish halo bordered by a ring of pigmentation (double-ring sign) ([picture 1](#)).
- Neck and chest
 - Goiter – Hypothyroidism.
 - Webbed neck, shield chest ([picture 2](#)) – Characteristic findings in Turner syndrome.
 - Webbed neck, pectus excavatum – Seen in Noonan syndrome.

- Suprascapular fat pad (buffalo hump) and supraclavicular fat pads – Suggests Cushing's syndrome. Mild forms of this fat distribution are seen in simple obesity (sometimes termed "pseudo-Cushingoid"), but exogenously obese children are often of normal or slightly increased stature.
- Limbs
 - Cubitus valgus (increased carrying angle of the arm), genu valgum – Commonly seen in Turner syndrome or **Short Stature H**omeobo**X** (SHOX) mutations
 - Madelung deformity of the forearm (focal dysplasia of the distal radial physis, leading to a prominent ulna and wrist pain) ([picture 3](#) and [image 1](#)) – Commonly seen in Turner syndrome or SHOX mutations
 - Stocky build – Often seen in Turner syndrome or SHOX mutations
 - Long limbs compared with trunk – Spondyloepiphysial dysplasia
 - Short limbs (especially upper arms) compared with trunk – Achondroplasia
 - Trident hands (broad, space between middle fingers) – Achondroplasia
- Skin – Atrophied skin, purple-colored striae are characteristic of Cushing's syndrome, sometimes with hyperpigmentation.
- Delayed or accelerated puberty, or hypogonadism – Most women with Turner syndrome have absent breast development; some have delayed puberty. Hypothyroidism tends to cause pubertal delay although, on rare occasion, this disorder may present with precocious puberty. Early puberty is occasionally seen in congenital virilizing adrenal hyperplasia or Cushing's disease. Microphallus or cryptorchidism suggests central hypothalamic-pituitary deficiencies.
- Decreased deep tendon reflexes – Suggests hypothyroidism.

LABORATORY AND IMAGING STUDIES — There is some controversy about the extent of testing that should be performed for children and adolescents with presumed idiopathic short stature (ISS). In otherwise healthy children, the yield of testing is extremely low. One study described the clinical evaluation of 235 children who were referred for short stature and had height <3rd percentile, but normal growth rate and no symptoms [16]. In this group of low-risk short children, nearly 99 percent were ultimately diagnosed with variants of normal growth (23 percent with familial short stature, 41 percent with constitutional delay of growth, and 36 percent with ISS). The cost per new diagnosis identified was more than \$100,000, although most patients did not undergo all of the screening tests that have been recommended in consensus guidelines [9]. Because of the low diagnostic yield and high cost, we suggest performing laboratory testing only selectively in otherwise asymptomatic children, as outlined below.

- A basic evaluation** consisting of a bone age determination is appropriate for children with short stature, normal growth rate (eg, height velocity [HV] at least 5 cm/year between four and six years of age, and at least 4 cm/year between six years and puberty), and no other symptoms. The bone age determination provides a more accurate assessment of adult height and clarifies the type of growth defect, as described above (see '[Is there evidence of delayed or accelerated growth?](#)' above). Screening for celiac disease is also reasonable for children with gastrointestinal symptoms or first-degree relatives with celiac disease.
- Broader testing** may be warranted if the child is severely short (eg, height \leq -2.5 standard deviations [SD, 0.6th percentile]), has growth failure (eg, height-for-age curve crossing two major percentile lines, or HV <5 cm/year between four and six years of age, and <4 cm/year between six years and puberty), or if the history or physical examination raised

suspicion for a specific systemic, endocrine, or genetic disorder. Decisions regarding the extent of testing should be made in conjunction with a pediatric subspecialist, but vary with the child's presenting symptoms and the clinical setting. We perform the following battery of tests to screen for several pathologic causes of short stature ([table 1](#)), as outlined in a consensus statement [\[9\]](#):

- Complete blood count and erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP).
- Electrolytes, creatinine, bicarbonate, calcium, phosphate, alkaline phosphatase, albumin.
- Celiac serologies (eg, tissue transglutaminase [tTG] immunoglobulin A [IgA] and total IgA) – Several guidelines recommend celiac screening for short stature, as well as for other indications. This test is relatively cost effective and screens for a common disease. Some celiac disease test panels include a measurement of total IgA, to exclude the unlikely possibility of a false-negative test in a patient with selective IgA deficiency. (See "[Diagnosis of celiac disease in children](#)", section on 'Whom to test'.)
- Thyroid stimulating hormone (TSH), free thyroxine (T4), insulin-like growth factor-I (IGF-I), and insulin-like growth factor binding protein 3 (IGFBP-3). IGFBP-3 has higher sensitivity for predicting the diagnosis of growth hormone deficiency in children <10 years of age as compared with IGF-I.
- Karyotype (in all girls, to rule out Turner syndrome, and in boys with associated genital abnormalities).
- Morning luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in children with signs of sexual precocity ("ultrasensitive" assays should be used for pediatric patients, and samples should be drawn in the early morning for optimal sensitivity). (See "[Definition, etiology, and evaluation of precocious puberty](#)".)

Additional testing for specific disorders is appropriate if the history and physical examination suggest a particular diagnosis, such as precocious puberty or an endocrine, skeletal, or syndromic cause of growth failure, as described above. (See "[Are there features suggesting pathologic growth failure?](#)" above and "[Causes of short stature](#)", section on 'Pathologic causes of growth failure'.)

Patients with reduced HV and low IGF-I and/or IGFBP-3 and delayed bone age should be evaluated for growth hormone deficiency using provocative tests. (See "[Diagnosis of growth hormone deficiency in children](#)".)

Contrast-enhanced magnetic resonance imaging (MRI) of the brain is appropriate for children with established growth hormone deficiency, or in those with signs or symptoms suggesting hypothalamic-pituitary dysfunction (eg, hypoglycemia, microphallus, cryptorchidism, septo-optic dysplasia, intracranial tumor, or history of cranial irradiation).

INFORMATION FOR PATIENTS — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see ["Patient information: My child is short \(The Basics\)"](#))

SUMMARY AND RECOMMENDATIONS

- Short stature is defined as height that is 2 standard deviations (SD) or more below the mean height for children of that sex and chronological age in a given population. This translates to a height that is below the 2.3rd percentile. The clinical significance of the short stature depends on many factors, including genetic potential and changes in stature over time (growth velocity). (See ["Is the child short?"](#) above.)

- Determination of the child's growth rate, or height velocity (HV), is an essential component of the evaluation for short stature. For children two years and older, growth failure is suggested by a growth pattern that has deviated downwards across two major height percentile curves, or by growth slower than the following rates:

- Age two to four years: HV less than 5.5 cm/year (<2.2 inches/year)
- Age four to six years: HV less than 5 cm/year (<2 inches/year)
- Age six years to puberty:
 - HV less than 4 cm/year for boys (<1.6 inches/year)
 - HV less than 4.5 cm/year for girls (<1.8 inches/year)

(See ["Is the child's height velocity impaired?"](#) above.)

- An estimate of a child's adult height potential can be obtained by calculation of the midparental height (target height), adjusted for the sex of the child ([calculator 4](#)). For boys and girls, 8.5 cm (2.4 inches) on either side of this calculated value represents the 3rd to 97th percentiles for anticipated adult height. (See ["Is the child's growth within the range for the family?"](#) above.)

- The history and physical examination should include (see ["Additional evaluation for causes of short stature"](#) above):

- Family history of growth and pubertal onset
- Review of systems for features suggestive of gastrointestinal, pulmonary, immunologic, or other systemic disease
- Dysmorphic features, especially webbed neck, cubitus valgus, and absent puberty in girls (suggests Turner syndrome), or disproportionate short stature (ie, short limbs compared with trunk).

- The two most common causes of short stature are familial (genetic) short stature and constitutional delay of growth and puberty (CDGP), which are normal variants of growth. These growth patterns often can be distinguished from one another, but some children have features of both ([table 2](#)). (See ["Are there features that suggest that this is a normal variant of short stature?"](#) above.)

- Important pathologic causes of growth failure that may present with short stature and/or delayed puberty include Crohn disease, celiac disease, and Turner syndrome ([table 1](#)). (See ["Are there features suggesting pathologic growth failure?"](#) above.)

- Laboratory evaluation for a child with short stature depends on the results of the above evaluation ([algorithm 1](#)).

- For children with short stature, normal growth rate (eg, HV at least 5 cm/year between four and six years of age, and at least 4 cm/year between six years and puberty) and no other symptoms, we suggest a basic evaluation including bone age determination. The bone age result can then be used to refine the estimate for the child's adult height and also informs the evaluation for possible causes of short stature. (See '[Bone age determination](#)' above and '[Laboratory and imaging studies](#)' above.)
- Children with severe short stature (eg, height \leq -2.5 SD [0.6th percentile]) or growth failure should be further evaluated with a complete blood count (CBC), erythrocyte sedimentation rate (ESR), tissue transglutaminase (tTG) immunoglobulin A (IgA), creatinine, electrolytes, thyroid stimulating hormone (TSH), free thyroxine (T4), insulin-like growth factor-I (IGF-I), and insulin-like growth factor binding protein-3 (IGFBP-3). A karyotype should be performed in all girls with unexplained short stature (to evaluate for Turner syndrome), and in short boys with associated genital abnormalities. Abnormal results of these tests and/or symptoms are signs a disorder causing growth failure warrants further investigation. (See '[Laboratory and imaging studies](#)' above and '[Causes of short stature](#)', section on 'Endocrine causes of growth failure'.)

Use of UpToDate is subject to the [Subscription and License Agreement](#).

REFERENCES

1. [Grimberg A, Kutikov JK, Cucchiara AJ. Sex differences in patients referred for evaluation of poor growth. J Pediatr 2005; 146:212.](#)
2. August, GP, Lippe, BM, Blethen, SL. et al. Growth hormone treatment in the United States demographic and diagnostic features of 2331 children. In: National cooperative growth study (NCGS) advisory group, Growth Hormone: Science, Research, and the NCGS: 10 years of research, Gardiner-Caldwell SynerMedCalifon, NJ, 1996. p. 179.
3. Chatelain P. Trends in the diagnosis and treatment of short stature as revealed by KIGS. In: Growth Hormone Therapy in KIGS: 10 Years' Experience, Ranke, MB, Wilton, P (Eds), Johann Ambrosius Barth Verlag, Heidelberg, 1999. p. 11.
4. [Grimberg A, Feemster KA, Pati S, et al. Medically underserved girls receive less evaluation for short stature. Pediatrics 2011; 127:696.](#)
5. [Karlberg J. A biologically-oriented mathematical model \(ICP\) for human growth. Acta Paediatr Scand Suppl 1989; 350:70.](#)
6. [HEALY MJ, LOCKHART RD, MACKENZIE JD, et al. Aberdeen growth study. I. The prediction of adult body measurements from measurements taken each year from birth to 5 years. Arch Dis Child 1956; 31:372.](#)
7. [Kerrigan JR, Rogol AD. The impact of gonadal steroid hormone action on growth hormone secretion during childhood and adolescence. Endocr Rev 1992; 13:281.](#)
8. [Tanner JM, Davies PS. Clinical longitudinal standards for height and height velocity for North American children. J Pediatr 1985; 107:317.](#)
9. [Cohen P, Rogol AD, Deal CL, et al. Consensus statement on the diagnosis and treatment of children with idiopathic short stature: a summary of the Growth Hormone Research Society, the Lawson Wilkins Pediatric Endocrine Society, and the European Society for Paediatric Endocrinology Workshop. J Clin Endocrinol Metab 2008; 93:4210.](#)
10. [Tanner JM, Goldstein H, Whitehouse RH. Standards for children's height at ages 2-9 years allowing for heights of parents. Arch Dis Child 1970; 45:755.](#)
11. Greulich WW, Pyle SI. Radiographic atlas of skeletal development of the hand and wrist, Stanford University Press, Stanford 1976.
12. Tanner JM, Whitehouse RH, Cameron N, et al. Assessment of Skeletal Maturity and Prediction of Adult Height (TW2 Method), Academic Press, London 1983.
13. [BAYLEY N, PINNEAU SR. Tables for predicting adult height from skeletal age: revised for use with the Greulich-Pyle hand standards. J Pediatr 1952; 40:423.](#)
14. [Roemmich, JN, Blizzard, RM, Peddada, SD, et al. Longitudinal assessment of hormonal and physical alterations during normal puberty in boys. IV: Predictions of adult height by the Bayley Pinneau, Roche-Warner-Thissen & Tanner-Whitehouse methods compared. Am J Hum Biol 1997; 9:371.](#)
15. [Topor LS, Feldman HA, Bauchner H, Cohen LE. Variation in methods of predicting adult height for children with idiopathic short stature. Pediatrics 2010; 126:938.](#)
16. [Sisley S, Trujillo MV, Khoury J, Backeljauw P. Low incidence of pathology detection and high cost of screening in the evaluation of asymptomatic short children. J Pediatr 2013; 163:1045.](#)